The incidence of primary glomerulonephritis worldwide: a systematic review of the literature

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SHORT SUMMARY

A systematic literature review of the incidence of primary glomerulonephritis is presented. 40 papers published between 1980 and 2010 were critically appraised and included in the review. Overall, incidence was found to be between 0.2 and 2.5 /100 000/year in adults with lower incidence rates in children, for most types of glomerulonephritis.

ABSTRACT

Background

Little is known about the worldwide variation in incidence of primary glomerulonephritis. The objective of this review is to critically appraise studies of incidence published 1980-2010 so that an overall view of trends of these diseases can be found. This will provide important information for determining changes in rates and understanding variations between countries.

Methods

All relevant papers found through searches of Medline, Embase and ScienceDirect were critically appraised and an assessment was made of the reliability of the reported incidence data.

Results

This review includes 40 studies of incidence of primary glomerulonephritis from Europe, North and South America, Canada, Australasia and the Middle East. Rates for the individual types of disease were found to be in adults, 0.2/100 000/year for membrano-proliferative glomerulonephritis, 0.2/100 000/year for mesangio-proliferative glomerulonephritis, 0.6/100 000/year for minimal change disease, 0.8/100 000/year for focal segmental glomerulosclerosis, 1.2/100 000/year for membranous nephropathy and 2.5/100 000/year for IgA nephropathy. Rates were lower in children at around 0.1/100 000/year with the exception of minimal change disease where incidence was reported to be 2.0/100 000/year in Caucasian children with higher rates in Arabian children (9.2/100 000/year) and Asian children (6.2-15.6/100 000/year).

Conclusions

This study found that incidence rates of primary glomerulonephritis vary between 0.2/100 000/year and 2.5/100 000/year. The incidence of IgA nephropathy is at least 2.5/100 000/year in adults: this disease can exist subclinically and is therefore only detected by chance in some patients. In addition, referral policies for diagnostic biopsy vary between countries. This will affect the incidence rates found.

INTRODUCTION

Although much is known about clinical characteristics and natural history of the primary glomerulopathies, very little information on the epidemiology of these diseases is available from reviews. Insight into the baseline incidence of glomerulonephritis throughout the world can provide important information on trends of disease occurrence by sex, age and geographical location. New vaccines are being introduced and concerns have been raised about the potential associated risk of autoimmune diseases (46,47). It is therefore of interest to know what the baseline incidence rates across the world are so that concerns about possibly associated increased incidence rates of autoimmune diseases, such as glomerulonephritis, can be evaluated.

To our knowledge no other systematic review of incidence of the most common of the primary glomerulopathies has been conducted in the last three decades. In this paper we perform a systematic review, critically appraising studies of incidence of primary glomerulonephritis throughout the world.

Method

Searches of the Medline, EMBASE and Science Direct databases (1980 – June 2007) were carried out using the search terms 'glomerulonephritis,' 'IgA nephropathy', 'membranous nephropathy', 'membranoproliferative glomerulonephritis', 'mesangial proliferative glomerulonephritis', 'minimal change disease', 'focal segmental glomerulosclerosis', 'postinfectious glomerulonephritis', 'idiopathic crescentic proliferative glomerulonephritis', 'ANCA-associated necrotising crescentic glomerulonephritis', 'anti-glomerular basement membrane disease', 'kidney disease', 'incidence', 'incid*' and 'epidemiology.' In Medline, the individual disease names were searched for as well as the term 'glomerulonephritis' because this MeSH term does not include all types of glomerulonephritis as daughter terms in its hierarchical structure.

The inclusion criteria were that the studies reported original work, that the study reported incidence of specific forms of glomerulonephritis with reference to a denominator population, that the estimates of population size and person-time contributed were accurate and that efforts had been made to ascertain all incident cases. When assessing the likelihood of missing incident cases, papers were evaluated as follows: 1) for case finding studies, did the authors ensure that all of the subjects contributing to incidence denominator data would have been eligible to have the disease diagnosed and did the authors check all relevant medical records? 2) For all studies, were cases checked to ensure that they were incident and not prevalent? 3) For all studies, did the authors ensure that the cause of glomerulonephritis was autoimmune and not secondary to another disease? Where possible we only included incidence rates for cases of glomerulonephritis caused by autoimmunity, determination of which relied on information given in the paper.

The titles and abstracts of all of the studies produced by the searches were reviewed and those papers accepted for inclusion in the study were appraised. Studies published in English, French, German, Spanish or Dutch were included. Review papers identified were searched for secondary references reporting on original research; secondary references found from any of the other papers reviewed were also included.

A standard data abstraction form was used to record all details of the papers reviewed; a copy of this is given in the Appendix, figure 1A. Each study was scored for accuracy of the incidence rates it presented and was classified as being at low, medium or high risk for under- or over-estimation of reported incidence rates by considering the reliability of numerator and denominator data. For instance, inclusion of prevalent cases or those thought not to be caused by autoimmunity will have led to overestimated rates as will underestimated denominator data. Conversely, missing cases or an overestimated denominator (e.g. a catchment area from which not all inhabitants had access to hospital services) would be considered to have resulted in underestimated incidence rates. Explanations provided by the papers' authors as to why incidence rates

were as expected or whether they were considered to be an over- or underestimate of the true incidence rate were taken into account in this process. If the extent of likely error was considered to be very great, the study was excluded. To minimise subjectivity, this assessment was agreed between two of the authors and random checks were performed to ensure consistency. Rates are presented as the number of cases/100 000/year and where sufficient data were given in the paper, rates were checked for accuracy. Guidelines were followed in the reporting of this study to ensure that key information was presented (1).

Results

The results of the database searches with the number of included and excluded papers are given in figure 1; the excluded references are available on request from the authors.

Most of the papers rejected at abstract review stage did not report on primary and autoimmune glomerulonephritis and had been found from the search using 'kidney disease' as the search term. Of the remaining papers, reasons for rejecting included those reporting on an ill sub-group of the population (e.g. those with systemic lupus erythematosus), those that reported on prevalence and not incidence, review papers and those that gave incidence rates as a percentage of people who had a renal biopsy.

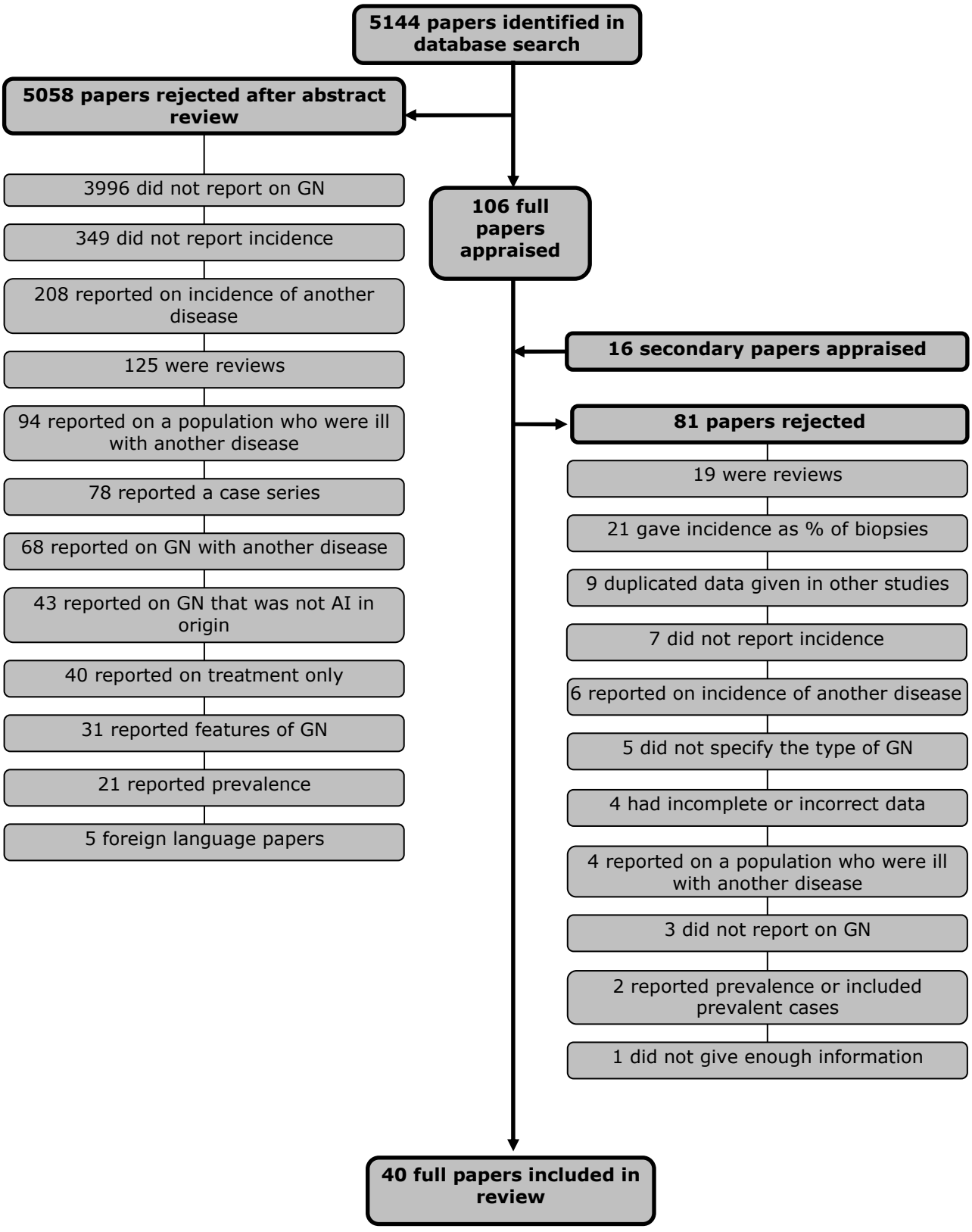

Figure 1: Results of the database searches showing the number of references found, those excluded and the final number of papers included in this review. (GN = glomerulonephritis; AI = autoimmune)

For some types of glomerulonephritis, the terms used by authors varied, some using the term to describe clinical presentation or syndrome for example crescentic proliferative (2) or crescentic glomerulonephritis (3-5), nephrosis (2,6), rapidly progressive glomerulonephritis (7,8), acute glomerulonephritis (9) and acute nephritis (10) whereas others used non-specific terms such as vasculitis (11-15) and total glomerulonephritis (9). These incidence rates have been excluded from the review because it was impossible to determine the extent to which these papers reported on primary and autoimmune glomerulonephritis.

Descriptions of the studies are given in table 1 and incidence rates are displayed in forest plots showing rates for children in figure 2 and adults or all ages in figure 3. Appendix 1, table 1A includes a table of all rates presented in the forest plots. Most of the studies included in this review investigated populations in Australasia (3,11,16), Europe (2,4-6,9,14,17-30), and North America (31-36) with five studies from the Middle East (13,37-40), two studies from South America (15,41), one study from Japan (42) and one study from Tunisia (43). Findings for the different subtypes of glomerulonephritis are summarised below.

IgA nephropathy: four studies reported rates in children and teenagers: 0.03 /100 000/year (CI_{95} 0-0.1) in Venezuela (41), 0.08 (CI_{95} 0.01-0.46) /100 000/ year (1975-1984) to 0.57 (CI_{95} 0.23-1.18) /100 000/ year (1985-1994) (0-17 years) in Tennessee, US (33), 4.5 /100 000/year (0-15 years) in Japan (42) and 0.31 /100 000/year (0-15 years) in Italy (25) (CI_{95} indicates 95% confidence interval). The difference in these rates may be explained by the study type: the Japanese study was a screening study and therefore detected subclinical cases of IgA nephropathy as well as including three possibly prevalent cases (total 37) who did not have previous screening results recorded. In contrast, the studies from Tennessee and Italy were based on renal biopsy and the study from Venezuela used chart reviews of symptoms and some biopsy results.

Most of the other studies were prospective, reporting rates from 0.2 /100 000 /year to 2.8 /100 000/year (2-6,13,18,19,21,27,43); the retrospective studies reported a similar range of rates, from 0.4 /100 000/year to 2.9 /100 000/year (9,11,14,20,29,30,35,36,38,39). Two studies reported incidence rates of 5.0/100 000/year (28) and 5.7/100 000 /year in males (11) but both had high biopsy rates which is likely to have contributed to the greater incidence rates found. Many of the studies included in this review were conducted between 1970 and 1990; no trend in incidence rates with time was discernable. Sixteen studies (3-6,9,11,13,14,20,21,25,27,29,32,35,38) reporting rates used immunofluorescence in diagnosing IgA nephropathy.

Membranous nephropathy: two studies (25,31) gave incidence rates in children and adolescents: 0.05 /100 000/year (1985-1993) to 0.09 /100 000/year (1993-2002) (6 months – 19 years) in Ottawa-Hull Canada (31) and 0.02 /100 000/year (0-15 years) in Italy (25). For the other studies, most were prospective and reported incidence rates between 0.3/100 000 /year and 1.4 /100 000/ year (2-6,13,18,19,21,27,28); the retrospective studies reported incidence to be between 0.2/100 000/year and 1.3/100 000/year (9,11,14,15,20,26,29,30,35,38,39,44). Not many studies reported on differences in rates between males and females. In those that did, the numbers were low and no confidence intervals or indications of statistical significance for differences in incidence rates were available. Three studies (6,9,11) suggested the rates were higher in males than females whereas El Reshaid *et al*. (13) reported the opposite. Taking into account the methods used in the studies presented, it is not thought that there has been a change in incidence between 1970 and 2000. The studies that used retrospectively collected data are likely to have missed cases and therefore underestimate the incidence rates. There was insufficient information to conclude reliably whether there is a difference in risk between males and females. Our best estimate of the incidence is 1.2 /100 000/year.

Membrano-proliferative glomerulonephritis: thirteen studies were retrospective and six were prospective; the range of incidence rates in all

studies was between 0.14 /100 000/year and 0.93 /100 000/year. Over time, incidence appears to have decreased from around 0.7 /100 000/year in the 1970s (3,6,18) to 0.2 in the 1990s (2,5,9,14) with the exception of Covic *et al* (26) who report a rate of 0.93/100 000/year in 2004. Covic *et al* (26) link this to the higher rates of streptococcal infection and hepatitis B and C in their population; they also note a decrease in the prevalence of membranoproliferative glomerulonephritis between 1995 and 2004 which they believe is associated with improvements in income, sanitation, social and medical infrastructure. Simon *et al.* (2,6) noted that there was an association between streptococcal infection and the onset of membrano-proliferative glomerulonephritis and that the decreases in incidence rates of membrano-proliferative and post-streptococcal glomerulonephritis were closely linked. Other differences in rates are noticeable: Hachicha *et al.* (43) found the highest rate of membrano-proliferative glomerulonephritis although this is probably an over-estimate of the true rate; in New Zealand (3) an incidence rate five times higher in Polynesians than in non-Polynesians was reported. This may indicate an infection related glomerular-specific injury. Two studies (2,3) reported on the incidence of type I membrano-proliferative glomerulonephritis, two studies (9,14) reported that 68% of their cases were type I and in a third study (30) 90% were type I; the remaining studies (3,5,6,11,18,19,25-27,31,38,39,45) did not specify the type of membrano-proliferative glomerulonephritis diagnosed.

Mesangial proliferative glomerulonephritis : four studies included were prospective and three were retrospective; all gave incidence rates between 0.2 /100 000/year and 1.1/100 000/year (3,5,6,26,27,44). In order to differentiate mesangial proliferative glomerulonephritis from IgA nephropathy, immunofluorescence should be used. All seven studies used immunofluorescence in their studies but four reported that this was not performed on all biopsies (26,27,30,44) giving a potential overestimation of the incidence rate. The only study to make the distinction between IgA nephropathy and mesangial proliferative glomerulonephritis without IgA deposits was that by Schena *et al* (5), who found an incidence rate of

0.16/100,000/year therefore it is likely the 'true' incidence rate of mesangial proliferative glomerulonephritis is at the lower end of the range given.

Minimal-change disease: in children, minimal change disease has been found to cause over 75% of cases of nephrotic syndrome (46). Seven studies reported on incidence of nephrotic syndrome, which would produce a slight overestimation of incidence of minimal change disease (22,24,34,37,40,41); three studies reported on incidence of minimal change nephrotic syndrome (23,25,31). Incidence rates in children were between 0.23 /100 000/year and 15.6 /100 000/year (22-25,31,34,37,40,41). The rates were reported with respect to ethnic origin and differences were noted: 0.23 -2.8/100 000/year in Caucasian children (22,24,25,31); 2.4/100 000/year in Hispanic children (41); 3.4 /100 000/year in Afro-Caribbean children (22); 7.2-11.6/100 000/year in Arabian children (37,40) and 6.2-15.6/100 000/year in Asian children who resided in the UK (22,23).

In the remaining studies, retrospective and prospective studies reported similar rates between 0.2/100 000/year and 0.8/100 000/year in adults (3,5,9,11,15,18,21,26,27,29,30,35,38,39,44); no trend of changes over time was found. Taking into account the accuracy of these rates, our best estimate of incidence of minimal change disease in adults is 0.6/100 000/year.

Focal segmental glomerulosclerosis: most rates, whether from prospective or retrospective studies, were between 0.2 /100 000/year and 1.1 /100 000/year; the Australian study reported the highest rates of 2.5/100 000/year in males and 1.8/100 000/year in females. The latter may be due to the fact that in Australia, people are referred for biopsy more often than in other countries included in this review (11). In the three studies that investigated differences in rates between males and females, incidence appeared to be higher in males (9,11,13). However no indication was given of the statistical significance of these differences and

given the fact that the numbers of cases were low, they may have arisen by chance.

Other types of glomerulonephritis: Incidence rates were presented for other types of glomerulonephritis, however these were limited to just one or two studies per disease type. Details are given in the forest plots: generally these rates were low at less than 1.0 /100 000/year. However Becquet *et al.* (16) in their study of post-infectious glomerulonephritis in French Polynesia, reported an incidence rate of 18/100 000/year; this is likely to be an underestimate of the true rate. This country had a higher rate of bacterial infections due to the climatic conditions, greater numbers of people sharing residences, low socioeconomic level and a lower use of medical care due to cultural beliefs; these factors are all thought to contribute to this higher incidence rate.

Accuracy of incidence rates

There are a number of components to assess when considering the incidence rates presented. Indication of likely accuracy of rates has been given in table 1. Of key importance is whether the cases included in the numerator were new cases: six studies reported in their methods section that only new cases were included (2,4,6,9,22,24,36,37). As glomerulonephritis is diagnosed by biopsy, the more liberal the biopsy policy, the greater the possibility of detecting all cases of the disease. Some studies gave their biopsy rate per head of population: these are given in table 1 and vary between 1.08 and 24.7 /100 000/year (6,11,14,18,19,21,26-30,35,38,39,44).

Discussion

This literature review found incidence rates for different types of primary autoimmune glomerulonephritis to be between 0.2/100 000/year and 2.5/100 000/year in adults. Most studies were from the US and France therefore it is difficult to draw conclusions regarding variability of rates with geographical location or ethnicity. Given that glomerulonephritis can exist subclinically, and given differences in access to renal biopsy between different healthcare systems, it is likely that geographical variations in

incidence rates found can be explained by differences in diagnosis rather than by genuine difference in disease frequency.

It is useful to note the type of studies undertaken to determine incidence of glomerulonephritis: in reviews of incidence of other autoimmune diseases (47,48), prospective studies are thought to have given more accurate rates than retrospective studies. However, in the studies presented here the rates reported were consistent irrespective of the study type.

Most studies used biopsy to diagnose the disease for the majority of cases however only five studies (3,21,27,36,40) reported the guidelines used for diagnosis. Classification of the disease and detection threshold of signs and symptoms may cause variation in incidence rates between studies (11,14,21) and the lack of a central histopathology review may have caused within-study variations (14). Wyatt *et al.* (32) reported that over time there was an increased recognition of IgA nephropathy therefore the incidence rates from the end of the study may be more accurate. Similarly Mazzuchi *et al* (15) reported rates using a national registry and found an increase in incidence with time they reported to be due to a greater awareness of the disease and earlier diagnosis.

Cases of IgA nephropathy can exist subclinically and therefore will only be diagnosed through routine urinary tests or if a patient presents with severe symptoms (27): Simon *et al.* (6) reported that 60% of cases of idiopathic IgA nephropathy were discovered by chance through routine testing as part of a medical examination in employment. Screening populations for conditions that can exist subclinically will produce higher and more accurate incidence rates especially when done routinely so that prevalent cases are not included in the incidence rate. In this review one study (42) used a regular screening program to find cases and the rate produced was greater by nearly an order of magnitude than other comparable incidence rates. Sehic *et al.* (33) reported that many cases of IgA are never diagnosed and that limited access to medical care for those

of lower socio-economic status may explain some failure to diagnose in this study.

There is the possibility of cases of secondary glomerulonephritis being included as primary cases particularly in retrospective studies; this would lead to an overestimation of incidence rates. There are links, for example between membrano-proliferative glomerulonephritis and hepatitis B or hepatitis C. Variations between countries in terms of rates of infections will contribute to differences in incidence rates.

One of the key factors in explaining differences in incidence rates of glomerulonephritis is the difference in referral and biopsy policies between different countries and even between regions of countries (14,21,28,29). Briganti *et al.* (11) found higher incidence rates of minimal change disease and focal segmental glomerulosclerosis than other studies. They claim this may be as a result of a more liberal biopsy policy, leading to the detection of less well-defined or asymptomatic cases. In contrast, The New Zealand Glomerulonephritis Study (3) noted that children and teenagers with postinfectious GN or minimal change nephropathy are rarely biopsied and older patients may also be less likely to have biopsies taken than younger adults. There is evidence that Polynesians have poorer access to healthcare resources in New Zealand than others and in this study, a high proportion of Polynesian patients presented for the first time with end-stage renal failure, making it impossible to reach a specific diagnosis. Lack of access to healthcare may also have resulted in prevalent cases being included in the incidence rates for glomerulonephritis in this population. Naumovic *et al* (30) reported that their low biopsy rate (1.08/100 000/year) may be due to economic sanctions and that elderly people and those with diabetes do not routinely undergo biopsy. Incidence was found to be lower in African-American children compared to Caucasian children (33). The study authors suggested this may be because the decision to perform a biopsy for those suspected to have mild disease was referred to their parents who may have been less likely than Caucasian parents to give their consent; records were not available for the number of biopsies refused (17).

Renal biopsy policy also affects diagnosis rates in the elderly: a number of studies reported increases in the number of elderly patients biopsied as a result of a change in policy and increasing age of the population (2,4,9,32) with Stratta *et al*. (9) reporting that rates of elderly patients diagnosed increased from 1.6% in 1970 to 20.4% in 1994. Wyatt *et al.* reported an increase in incidence at 45 years and over which was thought to be due to a more proactive attitude towards conducting diagnostic procedures in the elderly. It is likely that incidence in older people is underestimated as not all cases were referred to specialists or underwent biopsies (9).

Conclusion

Reported incidence rates of glomerulonephritis in adults varied between 0.2 and 2.5 /100 000 /year depending on the type of glomerulonephritis. Incidence in children was generally lower with most rates around 0.1 /100 000 /year; two exceptions to this were that incidence of minimal change disease in children was around 2.0 /100 000/year and a screening study that reported a rate of IgA nephropathy of 4.5 /100 000 /year in children in Japan.

The reported incidence rates are likely to underestimate true rates of IgA nephropathy as this disease can exist subclinically and may never be detected however other types of glomerulonephritis may be overestimated due to relapses and prevalent cases being counted as incident. There is variation in biopsy policy between countries, which affects the incidence rates found. Incidence in older people appears to have increased over time: this is considered to be due to greater inclusion of this age group in referrals for biopsy rather than due to a genuine increase in disease occurrence.

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Table 1: Details of diagnosis of cases and population covered for each of the studies included in this report

Study	Cases	RUE	ROE	Biopsy rate	Description
Hachicha *et al*. (43) **Study type:** Retrospective hospital record review **Location:** Sfax, Tunisia	611	**	***	Not given	**Case definition:** >14 years. Renal biopsy in 230 cases; histological disease type determined. Very few study details given. **Case identification:** No details given.
Utsunomiya *et al*. (42) **Study type:** Prospective screening study **Location:** Yonago City, Japan	37	**	**	99.5%	**Case definition:** Initial screening of first urine of morning; second screening of those with proteinuria, occult blood and urinary sediment. Those with glucosuria were referred. Follow-up for a few months. **Case identification:** All students aged 6-15 years screened annually. Indication for renal biopsy determined according to criteria. Records of previous screenings checked to identify new cases.
Briganti *et al*. (11) **Study type:** Retrospective **Location:** Victoria, Australia	1147	*	*	21.5	**Case definition:** Renal biopsy. **Case identification:** Retrospective review of pathology reports of all biopsies; evaluated by light microscopy and immunofluorescence or immunohistochemistry; majority assessed by electron microscopy. No details available of clinical findings or indication for biopsy.
The New Zealand Glomerulonephritis Study (3) **Study type:** Prospective **Location:** New Zealand	803	***	*	'Liberal'	**Case definition:** > 14 years. Renal biopsy material examined using light, immunofluorescence and electron microscopy. Uniform histological classification agreed; all renal biopsies re-evaluated independently using Churg and Sobin classification. **Case identification:** 4 nephrology centres covering 75% of the Polynesian and 84% of the non-Polynesian populations used.

Study	Cases	RUE	ROE	Biopsy rate	Description
Becquet *et al*. (16) **Study type:** Retrospective **Location:** French Polynesia	12	*	*	Not given	**Case definition:** < 15 years. Diagnostic criteria were microscopic or macroscopic haematuria, decreased C3 fraction of the complement evidence of recent streptococcal infection established by presence of elevated anti-streptococcal antibody titres. **Case identification:** Admitted to one hospital.
Frimat *et al*. (17) **Study type:** Prospective **Location:** East France	Not given	*	*	Not given	**Case definition:** Prospective longitudinal cohort study; >15 years. Diagnoses based on renal biopsy and checked by pathologist. Study restricted to half geographical area to ensure all biopsies included**. Case identification:** 17 renal units.
Abdulmassih *et al*. (18) **Study type:** Retrospective **Location:** Picardy, France	266	**	* '76-80 '81-85	 7.2 8.6	**Case definition:** >15 years, diagnosed by biopsy. **Case identification:** All biopsies for the area examined in one lab.
Berthoux *et al*. (19) **Study type:** Prospective **Location:** Rhone-Alps, France	Not given	*	*	13.0	**Case definition:** Renal biopsy. **Case identification:** Sent questionnaire in 1989 to all nephrology services; also provided information for denominator.
Simon *et al*. (6) **Study type:** Prospective **Location:** St. Brieuc, France	480	*	* '76-80 '81-85 '86-90	 18.7 20.1 16.2	**Case definition:** Renal biopsy specimens processed and stained for light microscopy and immunohistory; electron microscopy not systematically performed. **Case identification:** Biopsy performed at hospital nephrology department; collaborated with major medical screening institutions.
Simon *et al*. (4) As above	131			Not given	**Case definition:** > 60 years old, otherwise, as above.
Simon *et al*. (2) As above	898			Not given	**Case definition and identification:** As above.

Study	Cases	RUE	ROE	Biopsy rate	Description
Simon *et al*. (45) As above	898			Not given	**Case definition and identification:** As above.
Schena *et al*. (5) **Study type:** Prospective **Location:** Italy	1293	**	**	~ 4.5	**Case definition:** Renal biopsies mainly evaluated by light-microscopy and immunofluorescence; electron-microscopy used in 38% of cases. **Case identification:** Biopsies collected at Italian renal units.
Coppo *et al.* (25) **Study type:** Prospective **Location:** Italy	256	**	**	Not given	**Case definition:** 0-15 years; renal biopsy analysed by light microscopy and immunofluorescence; electron microscopy used in 32% of cases. **Case identification:** Renal units where biopsies were performed.
Stratta *et al*. (9) **Study type:** Retrospective **Location:** City/ Province of Turin, Italy	454	*	*	Not given	**Case definition:** >15 years, renal biopsy taken during time period. Biopsies underwent light microscopy and immunofluorescence; electron microscopy not routinely used. **Case identification:** 3 nephrology centres in city (provided renal biopsies to virtually entire area); those outside region were excluded.
Rivera *et al*. (14) **Study type:** Retrospective **Location:** Spain	Not given	***	***	4.8	**Case definition:** Primary glomerulonephritis classified into eight groups; criteria not given. **Case identification:** Retrospective review of renal biopsies from national registry.
Grupo de Estudio de la Sociedad Espanola de Nefrologia (20) **Study type:** Retrospective **Location:** Spain	1471 (max.)	***	*	Not given	**Case definition:** Diagnosis established from kidney biopsies studied by light microscopy and immunofluorescence. >14 years. Membranoproliferative GN was classified as type I or type II Tried to identify all primary cases. **Case identification:** 33 hospitals responded.
Wirta *et al.* (28) **Study type:** Prospective	958	**	*	UH 24.7 CH 9.1	**Case definition:** Kidney biopsy; SNOMED classification **Case identification:** Patients receiving a renal biopsy at the university or central hospitals.

Study	Cases	RUE	ROE	Biopsy rate	Description
Location: Western Finland					
Heaf *et al.* (44) **Study type:** Retrospective **Location:** Denmark	1762	**	**	3.82	**Case definition:** Renal biopsy classified according to WHO guidelines and presence of immune deposits. **Case identification:** Renal biopsy register.
Tiebosch *et al*. (21) **Study type:** Prospective **Location:** Areas surrounding the cities of Heerlen, Maastricht and Sittard, The Netherlands	129	*	*	12.6	**Case definition:** Renal biopsies processed and stained for light microscopy, transmission electron microscopy and immunohistochemistry and classified according to WHO guidelines. **Case identification:** Biopsies taken in direct referral hospitals for GPs.
Hanko *et al*. (29) **Study type:** Retrospective **Location:** Northern Ireland, UK	907	*	* '76-85 '86-95 '96-05	 2.02 3.86 7.08	**Case definition:** > 16 years; all aduct native kidney biopsies analysed. **Case identification:** Renal services in pathology department at city hospital.
Sharples *et al*. (22) **Study type:** Retrospective **Location:** Birmingham, UK	44	**	***	Not given	**Case definition:** <16 years presenting with nephrotic syndrome responding to corticosteroids. Nephrotic syndrome defined as proteinuria of at least 3+ on Albustix testing with oedema and a plasma albumin concentration of 25 g/l or less. Steroid response defined as abolition of proteinuria within eight weeks of starting prednisolone. **Case identification:** Names and hospital numbers were traced for all admissions with the main or subsidiary diagnosis and responding to corticosteroid treatment.

Study	Cases	RUE	ROE	Biopsy rate	Description
Feehally *et al* (23) **Study type:** Retrospective **Location:** Leicestershire, UK	43	**	**	Not given	**Case definition:** <15 years**.** Minimal change nephrotic syndrome defined by complete abolition of proteinuria within four weeks in response to corticosteroids with no hypertension or renal impairment. Some had further information from biopsy material analysed by light, immunofluorescence and electron microscopy. **Case identification**. From hospital records.
McKinney *et al* (24) **Study type:** Retrospective **Location:** Yorkshire, UK	194	***	***	Not given	**Case definition:** 0-15 years. Nephrotic syndrome diagnosed if proteinuria was at least 3+ on testing with albustix, with hypoalbuminaemia and oedema. Investigated response to corticosteroids. **Case identification:** Primary: listings provided by paediatricians. Secondary: inpatient hospital episode statistics; identified those with ICD9 codes 580-583 (acute/chronic GN, NS, nephritis, nephropathy) 590 (infections of the kidney.) Data extracted from hospital notes. Ethnic group determined by surname.
Rychlik *et al* (27) **Study type:** Prospective **Location:** Czech Republic	1932	**	**	5.37	**Case definition:** Indications for biopsy differed between centres. Histological evaluation by light microscopy and immunofluorescence performed routinely, with electron microscopy in a number of cases. Histological classification used WHO recommendations. **Case identification:** Renal biopsy records collected from renal units. Questionnaire used to collect relevant data.
Covic *et al* (26) **Study type:** Retrospective **Location:** Moldova and Banat, Romania	Not given	**	**	11.3	**Case definition:** >18 years; diagnosed by renal biopsy. **Case identification:** Two large referral centres.

Study	Cases	RUE	ROE	Biopsy rate	Description
Naumovic *et al* (30) **Study type:** Retrospective **Location:** Serbia	Not given	***	**	1.08	**Case definition:** >18 years; diagnosed by renal biopsy; stained and analysed by light microscopy. **Case identification:** Nephrology unit.
Al Arrayed *et al* (38) **Study type:** Retrospective **Location:** Bahrain	218	**	**	5.8	**Case definition:** Indications for biopsy: proteinuria, unexplained microscopic or macroscopic haematuria, systemic disease with clinical evidence of renal involvement, unexplained renal impairment and renal impairment in post-transplant patients. **Case identification:** All renal biopsies, nephrectomy specimens and referral slides pertaining to renal disease reviewed.
Al Arrayed *et al* (39) As above	40	**	**	5.4	**Case definition:** As above. **Case identification:** As above.
El Reshaid *et al* (13) **Study type:** Prospective **Location:** Kuwait	315	*	***	Not given	**Case definition:** Histological diagnosis of biopsy made on results of light microscopy, immunofluorescence and electron-microscopy in selected patients. **Case identification:** Patients screened for glomerulopathy; those meeting criteria referred for biopsy.
Zaki *et al* (40) **Study type:** Prospective **Location:** Kuwait	55	**	**	Not given	**Case definition:** Patients had oedema, albuminuria and hypoalbuminaemia. Biopsy in those non-responsive to steroid medication over 4 weeks. International Society of Kidney Disease in Children guidelines used. **Case identification:** Children admitted to the paediatric hospital departments of in Kuwait.
Elzouki *et al* (37) **Study type:** Prospective **Location:** Benghazi, Libya	19	**	***	Not given	**Case definition:** <15 years. Renal biopsy performed in 17 of 19 cases. Standard methods of light and electron microscopy and immunofluorescence**. Case identification:** El-Fateh Children's Hospital and clinics in the area.
Filler *et al* (31) **Study type:** Retrospective	159	*	**	Not given	**Case definition:** Diagnosis made according to the International Study of Kidney Disease in Children criteria, verified by chart review. **Case identification:** All

Study	Cases	RUE	ROE	Biopsy rate	Description
Location: Ottawa-Hull region, Canada					inpatients and outpatients referred to nephrology services included. Hospital admission database and renal biopsy records also checked.
Wyatt *et al* (32) **Study type:** Prospective **Location:** Central and eastern Kentucky, US	192	**	*	Not given	**Case definition:** Renal biopsy using direct immunofluorescence; standard criteria. **Case identification:** Primary method of ascertainment by renal pathologists, hospital records also reviewed.
Swaminathan *et al* (35) **Study type:** Retrospective **Location:** Olmsted County, US	116	**	* '74-'83 '84-'93 '94-'03	 8.2 8.8 17.5	**Case definition:** Renal biopsy evaluated with light microscopy, immunofluorescence and electron microscopy; diagnosis confirmed by renal pathologist. **Case identification:** Record linkage gives details of virtually all medical care provided.
Fischer *et al.* (36) **Study type:** Retrospective **Location:** New Mexico	112	*	*	Not given	**Case definition:** WHO classification. **Case identification:** Kidney biospies from patients newly diagnosed with IgA at the university were retrieved from inhouse and consultation files.
Kim et al. (34) **Study type:** Retrospective **Location:** New Orleans	163	**	**	Not given	**Case definition:** Nephrotic syndrome defined as heavy proteinuria, edema and hypoalbuminemia; some underwent renal biopsy. **Case identification:** Record review and two main referral hospitals
Sehic *et al* (33) **Study type:** Retrospective **Location:** Shelby County, Tennessee, US	17	**	*	Not given	**Case definition:** Patients < 18 years and resident in study area. Diagnosis of IgA nephropathy made by renal biopsy.
Orta-Sibu *et al* (41) **Study type:** Retrospective **Location:** Venezuela	505	**	***	Not given	**Case definition:** <15 years; Acute GN with hematuria, edema, arterial hypertension present. Nephrotic syndrome was diagnosed on the basis of proteinuria greater than 40 mg/h per m^2 body surface area with or without edema,

Study	Cases	RUE	ROE	Biopsy rate	Description
					hypoproteinemia and hypercholesterolemia. **Case identification:** Information obtained by contacting 17 centres with a questionnaire. Data collected by chart review of patients.
Mazzuchi et al. (15) **Study type:** Prospective **Location:** Uruguay	2058	***	*	Not given	**Case definition:** Diagnosed by biopsy; defined by minimal glomerular lesions, levels of proteinuria, serum creatinine, arterial hypertension, glomerular filtration rate. **Case identification:** National registry

RUE: risk of underestimation; ROE: risk of overestimation; * low; ** medium; *** high; ESR: erythrocyte sedimentation rate; ANA: anti-nuclear antibodies; GBM: glomerular basement membrane; ELISA enzyme-linked immunosorbent assay; biopsy rate: /100 000/year

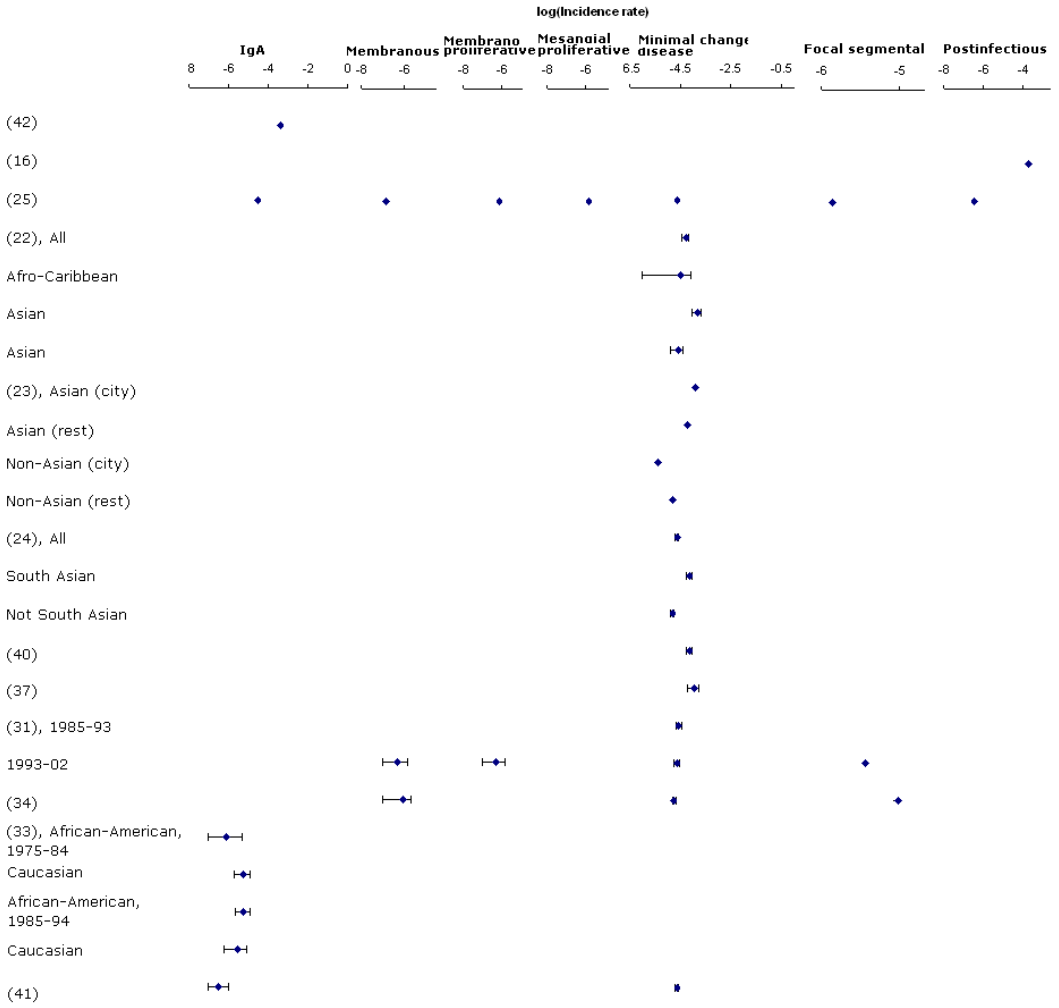


Figure 2: Incidence of glomerulonephritis in children and adolescents.

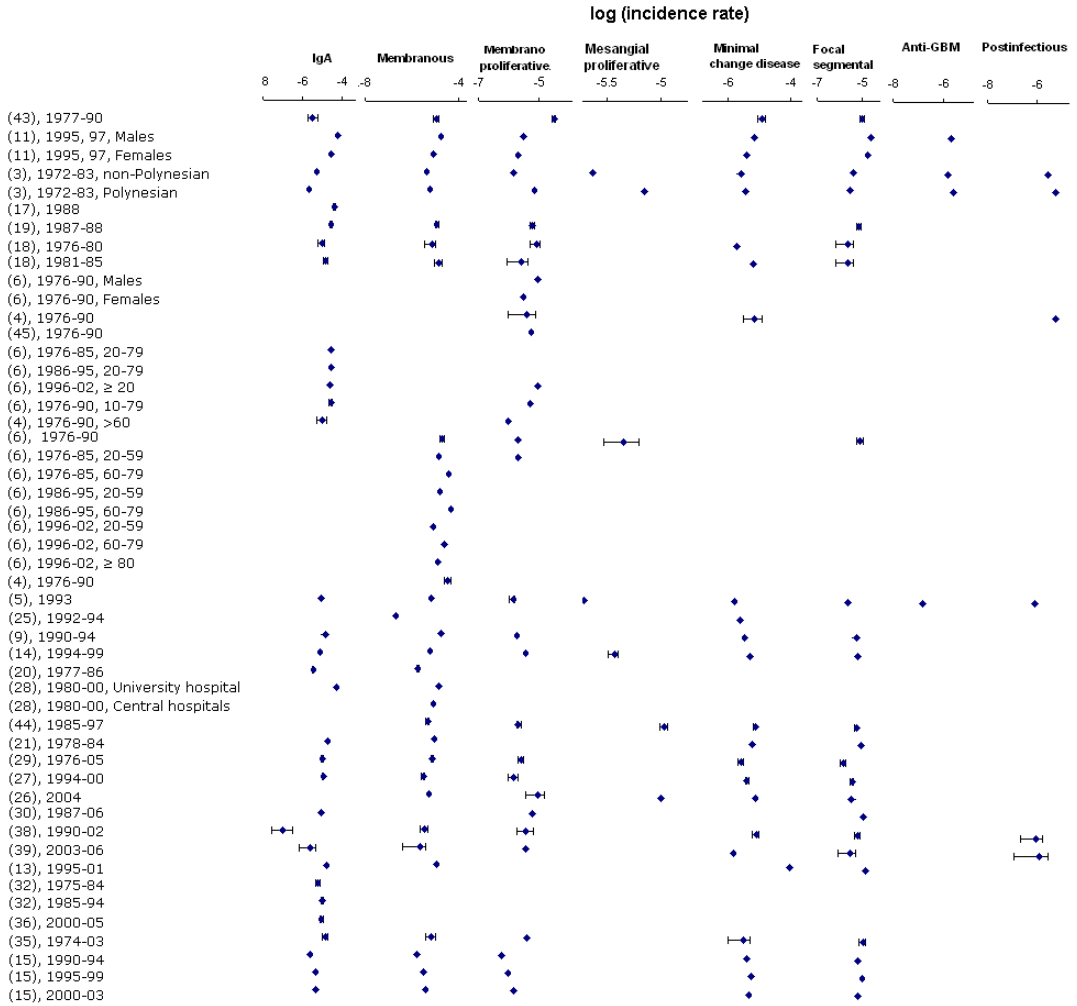
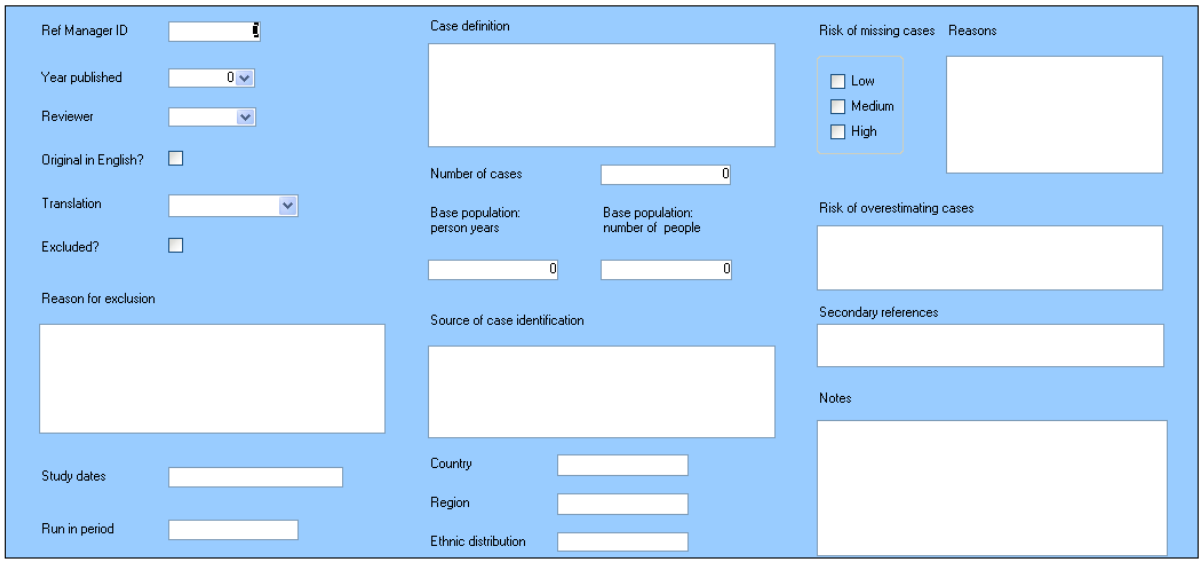


Figure 3: Incidence in adults or those of all ages

Appendix



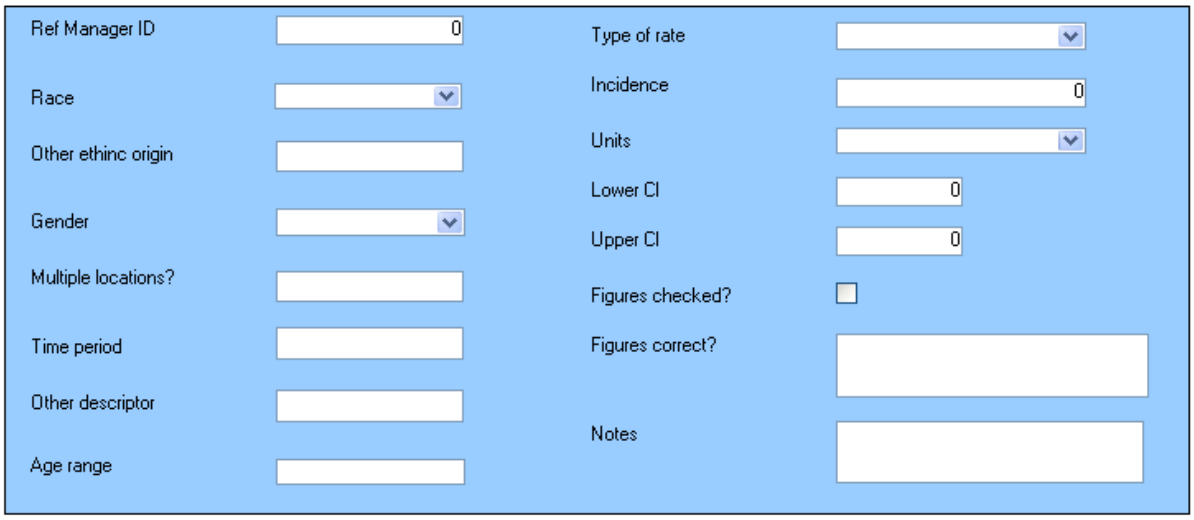


Figure 1a: Data abstraction forms

Table 1A: Incidence rates by type of glomerulonephritis; rates are crude and presented in units of /100 000 /year unless otherwise specified.

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
(43)	All	Sfax, Tunisia	1977-90	>14 years	0.3 (0.2, 0.6) §	1.1 (0.8, 1.4)§	3.1 (2.7, 3.6) §		1.2 (0.9, 1.5)§	1.0 (0.8, 1.3)§		
(42)	Asian	Yonago City, Japan	1983-1999	<15 years	4.5							
(11)	Caucasian	Victoria, Australia	1995, 97	All	M: 5.7, F: 2.9	M: 1.8, F: 0.8	M: 0.3, F: 0.2		M: 0.7, F: 0.4	M: 2.5, F: 1.8	M: 0.2, F: 0	
(3)	non-Polynesian Polynesian	New Zealand	1972-83	> 14 years	0.55‡ 0.22‡	0.43‡ 0.58‡	0.15‡ 0.74‡	0.23‡ 0.7‡	0.26‡ 0.35‡	0.42 ‡ 0.3 ‡	0.16‡ 0.25‡	0.26‡ 0.53‡
(16)	All	French Polynesia	2007	<15 years								18
(17)	Caucasian	East France	1988	> 15 years	4.02 (3.59, 4.46)¶							
(19)	Caucasian	Rhone-Alps, France	1987-88	All	2.7 (2.5, 3.0) ¶	1.2 (1.0, 1.4) ¶	0.6 (0.5, 0.7) ¶			0.7 (0.6, 0.9)¶		
(18)	Caucasian	Picardy, France	1976-80 1981-85	> 15 years	0.95 (0.60, 1.24) ¶ 1.51 (1.10,1.93	0.52 (0.26,0.74)¶ 1.03 (0.69,1.38) ¶	0.83 (0.50, 1.10) ¶ 0.26 (0.09,0.44) ¶		0.18 0.65	0.24 (0.07,0.40) ¶ 0.23 (0.00,0.19)		

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
					) ¶					¶		
(6)	Caucasian	Saint Brieuc, France	1976-85 1986-95 1996-2002	20-79 20-79 ≥ 20	2.8 2.8 2.6							
(6)	Caucasian	St. Brieuc, France	1976-90	0-79			M: 0.9, F: 0.3					
(6)	Caucasian	St. Brieuc, France	1976-90	10-79	M: 4.8, F: 1.4, B: 2.7 (2.2,3.0)¶	1.4 (1.1,1.7)¶		0.45 (0.29,0.62)¶		0.8 (0.6,1.1)¶		
(6)	Caucasian	Saint Brieuc, France	1976-85 1986-95 1996-2002 	20-59 60-79 20-59 60-79 20-59 60-79 ≥ 80		1.0 2.8 1.1 3.3 0.6 1.7 0.9						

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
(4)	Caucasian	St. Brieuc, France	1976-90	> 60 years	1.0 (0.5,1.6)¶	2.5 (1.7,3.3)¶	0.4 (0.1,0.8)¶		0.7 (0.3,1.2)¶			
(45)	Caucasian	St. Brieuc, France	1976-90	10-79			0.55					0.55
(2)	Caucasian	Saint Brieuc, France	1976-85 1986-95 1996-2002	20-59 60-79 20-59 60-79 20-59			0.9 0.5 0.1 0.2 0.2					
(5)	Caucasian	Italy	1993	All	0.84	0.49	0.14	0.19	0.16	0.23	0.01	0.07
(25)	Caucasian	Italy	1992-94	0-15	0.31	0.015	0.075	0.16	0.23	0.14		0.035
(9)	Caucasian	City or Province of Turin, Italy	1990-94	≥ 15	M: 2.27, F: 0.67, B: 1.47	M: 1.84, F: 0.79, B: 1.31	M: 0.24, F: 0.14, B: 0.19		M: 0.36, F: 0.27, B: 0.32	M: 0.62, F: 0.49, B: 0.55		
(28)	Caucasian	Western Finland	1980-2000	All	UH 5.0	UH 1.4 CH 0.8						

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
(44)	Caucasian	Denmark	1985-97	All		0.48 (0.4,0.51) ¶	0.21 (0.16,0.24)¶	1.08 (0.94,1.01)¶	0.73 (0.62,0.75)¶	0.57 (0.48,0.59)¶		
(21)	Caucasian	Heerlen, Maastricht and Sittard, The Netherlands	1978-84	16-65 years	1.9	0.9			0.6	0.9		
(14)	Caucasian	Spain	1994-99	All ages	0.79	0.62	0.36		0.48	0.64		
(20)	Caucasian	Spain	1977-86	> 14 years	0.35 (0.33,0.37) §	0.18 (0.17, 0.20)§						
(29)	All	Northern Ireland, UK	1976-05	>16 years	0.99 (0.88, 1.09) ¶	0.75 (0.66, 0.84) ¶	Type 1: 0.24 (0.19, 0.29) ¶ Type 2: 0.03 (0.01, 0.04) ¶		0.25 0.20, 0.30) ¶	0.15 (0.11, 0.18) ¶		
(23) Primary nephrotic syndrome	Asian (i.e. 11.9% of population) Non-Asian	Leicestershire, UK City Rest of country City Rest of	1973-82	0-15					12.1 6.2 0.4 1.6			

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
		country										
(24) Primary nephrotic syndrome	All South Asian Not South Asian	Yorkshire, UK	1987-98	0-15					2.3 (2.0-2.6) 7.4 (5.3-9.5) 1.6 (1.3-1.8)			
(22)	All Afro-Caribbean Asian Asian	Birmingham, UK	1979-83	0-16					5.3 (3.7, 7.0) ¶ 3.4 (0, 8.1) ¶ 15.6 (9.6, 21.9) ¶ 2.6 (1.3, 4.0) ¶			
(27)	Caucasian	Czech Republic	1994-2000	All	1.12 (1.04, 1.19)¶	0.30 (0.26, 0.34) ¶	0.15 (0.13, 0.18) ¶	0.37 (0.32, 0.40) ¶	0.40 (0.35, 0.45) ¶	0.35 (0.31, 0.39) ¶		
(26)	Caucasian	Moldova and Banat, Romania	2004	>18 years		0.53	0.93	1	0.73	0.33		
(30)	All	Serbia	1987-06	>18 years	0.85	1.24	0.61	1.08	0.19	1.11		

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
(38)	All	Bahrain	1990-2002	All	0.01 (0.00, 0.03) §	0.35 (0.23, 0.48)§	0.38 (0.25, 0.50)§		0.79 (0.60, 0.97)§	0.62 (0.45, 0.79) §		0.08 (0.02, 0.14)§
(39)	Arabian and non-Arabian	Bahrain	2003-06	All	0.26 (0.07,0.46) §	0.22 (0.04, 0.40)§	0.37 (0.14, 0.60)§		0.15 (0.00, 0.30)§	0.30 (0.09, 0.51)§		0.11 (0.00, 0.24) §
(13)	Kuwaiti national	Kuwait	1995-2001	All	M: 2.5, F: 1.0, B: 1.7	M: 0.8, F:1.4, B: 1.1			9.2	M: 1.8, F: 1.1, B: 1.4		
(40)	Kuwaiti national	Kuwait	1981-85	0-15					7.2 (5.3, 9.1)¶			
(37)	Arabian[1]	Benghazi, Libya	1980-82	0-15					11.6 (6.4, 16.9) ¶			
(31)	Caucasian	Ottawa-Hull region, Canada	1985-93 1993-2002	6 months - 19 years		0.05 (0, 0.14) ¶ 0.09 (0, 0.21) ¶	0.05 (0, 0.14) ¶ 0		2.81 (2.10, 3.52) ¶ 2.47 (1.81, 3.12) ¶	0.37 (0.11, 0.63) ¶ 0.94 (0.54, 1.34) ¶		
(32)	Caucasian	Central and Eastern Kentucky, US	1975-84	All	M: 0.92, F: 0.34, B: 0.62 (0.47,0.76)¶							

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
			1985-94		M: 1.43, F: 0.65, B: 1.02 (0.84,1.21)¶							
(35)	Caucasian	Olmsted County, US	1974-03	All	1.4 (1.0, 1.8)	0.7 (0.4, 1.0)	0.4 (0.2, 0.7)		0.3 (0.1, 0.5)	1.1 (0.7, 1.5)		
(36)	All	New Mexico	2000-05	All	0.93 (0.75. 1.11) ¶							
(34) Disease defined as nephrotic syndrome	Caucasian and African American	New Orleans, US	1994-03	1-18 years					1.81 (1.53, 2.09) ¶			
(33)	African-American Caucasian	Shelby County, Tennessee, US	1975-84	< 18 years	0.08 (0.01, 0.46) 0.56 (0.2 1.22)							

Ref	Race	Location	Time period	Age range	IgA nephropathy	Membranous nephropathy	Membranoproliferative GN	Mesangial proliferative GN	Minimal change disease	Focal segmental glomerulosclerosis	Anti-GBM disease	Postinfectious GN
	African-American Caucasian		1985-94		0.57 (0.23, 1.18) 0.3 (0.06, 0.87)							
(41)	All	Venezuela	1998	<15 years	0.03 (0, 0.1) §¶				2.4 (2.0, 2.7) §¶			
(15)	All	Uruguay	1990-94 1995-99	All	0.24 0.46 0.45	0.16 0.31 0.40	0.06 0.10 0.14		0.4 0.55 0.46	0.67 0.99 0.64		

Notes: ‡ incidence rate adjusted for age; § incidence rate calculated from data given in the paper; ¶ CI calculated from data given in paper using standard Normal distribution; ¹ Terminology as given in paper; UH University hospital; CH central hospital